Current Gastroenterology Reports (2022) 24:18–25
https://doi.org/10.1007/s11894-022-00839-4

NUTRITION AND OBESITY (S MCCLAVE AND E OMER, SECTION EDITORS)

When Can we Feed after a Gastrointestinal Bleed?

Rebecca A. Busch[1] · **Bryan R. Collier**[2] · **Matthew B. Kaspar**[3]

Accepted: 13 January 2022 / Published online: 11 February 2022
© The Author(s), under exclusive licence to Springer Science+Business Media, LLC, part of Springer Nature 2022

Abstract

Purpose of Review Gastrointestinal (GI) bleeding can carry minimal or significant risk for recurrent hemorrhage. Timing of feeding after GI bleeding remains an area of debate, and here we review the evidence supporting recommendations.

Recent Findings Improved understanding of the pathophysiology of GI bleeding and the evolution of treatment strategies has significantly altered the management of GI bleeding and the associated propensity for rebleeding. Early feeding following peptic ulcer bleeding remains ill-advised for high risk lesions while early initiation of liquid diets following cessation of esophageal variceal bleeding is appropriate and shortens hospital stays.

Summary Time to feeding following GI bleeding is inherently based on the disease etiology, severity, and risk of recurrent hemorrhage. With evolving standards of care, rates of rebleeding following endoscopic hemostasis are decreasing. Some evidence exists for early feeding however, larger multi-center trials are needed to help optimize timing of feeding in higher risk lesions.

Keywords Gastrointestinal bleeding · Feeding · Nutrition · Peptic ulcer disease · Esophageal variceal bleeding

Introduction

Over the last three decades we have witnessed significant evolution in the pathophysiology and management of gastrointestinal (GI) bleeding. With the advent of proton pump inhibitors, advancement in hemostatic techniques and increasing availability of advanced endoscopy the incidence of acute GI hemorrhage and its associated morbidity and mortality has decreased. Despite these significant gains in management, post-intervention care surrounding feeding after GI bleeding is still frequently managed by dogma. Here we aim to review current practice patterns and scientific updates surrounding feeding after management of GI hemorrhage.

Epidemiology and Etiology

GI bleeding can originate anywhere along the digestive tract. Clinically, GI bleeding is broadly classified as upper or lower GI bleeding with the ligament of Treitz acting as the anatomical divider. While many instances of GI bleeding are managed in the outpatient setting, overt GI bleeding with clinical evidence of hematemesis, hematochezia, or melena often prompts urgent or emergent evaluation. In the United States, hospitalization rates for upper GI bleeding are 67/100,000 population compared to 36/100,000 population for lower GI bleeding [1].

The etiology of GI bleeding and its associated risk of rebleeding, need for blood transfusion, and mortality guide further intervention and management. Certain types of bleeds

This article is part of the Topical Collection on *Nutrition and Obesity*

✉ Rebecca A. Busch
busch@surgery.wisc.edu

Bryan R. Collier
brcollier@carilionclinic.org

Matthew B. Kaspar
matthew.kaspar@vcuhealth.org

[1] Department of Surgery, Division of Acute Care and Regional General Surgery, University of Wisconsin- Madison, Madison, WI, USA

[2] Department of Surgery, Section of Acute Care Surgery, Virginia Technical Institute Carilion School of Medicine, Roanoke, VA, USA

[3] Department of Internal Medicine, Division of Gastroenterology, Hepatology, and Nutrition Virginia Commonwealth University Medical Center, Richmond, VA, USA

SHOUT
SHARE
IT

tend to be self-limited while others are life threatening. Risk stratification scores such as the Glasgow-Blatchford score for upper GI bleeds and Oakland score in the case of lower GI bleeds use patient demographics, physical exam findings, and laboratory values to identify patients at low risk of adverse outcomes who can be safely discharged from the emergency room. All other patients typically require further evaluation with endoscopy for diagnostic and often therapeutic intervention.

Time to Feeding

Optimal timing of feeding after evaluation and treatment of acute GI bleeding is a topic of much debate. Often endoscopic findings dictate post-procedure nutrition. Patients with GI bleeding associated with gastric erosions, gastritis, Mallory-Weiss tear, esophagitis, angiodysplasia or lower GI bleeding in general may resume feeding without delay as these bleeds tend to be self-limited and rarely require intervention [2–5]. For patients with upper GI bleeding associated with gastric or duodenal ulcers, post-procedure diet is frequently tied to the ulcer's risk of rebleeding based on the Forrest classification (Table 1). If patients have a Forrest IIc or III lesion (flat pigmented spot on the ulcer base or clean ulcer base, respectively) [6], it is relatively well accepted that they may resume a regular diet following esophagogastroduodenoscopy (EGD) [2, 5]. However, for patients with Forrest I-IIb ulcers (active spurter, active oozing, non-bleeding visible vessel, or adherent clot) or bleeding from esophageal or gastric varices post-procedure management regarding resuming nutrition is not well established. How to best manage nutrition in these patients has been the subject of clinical research since the early 1990s with only modest shifts in clinical care if any.

History of Fasting

Patients hospitalized with acute upper GI bleeds were historically kept nil per os (NPO) for 48 to 72 h after their index procedure in effort to reduce the risk of rebleeding. For ulcer disease, most high risk lesions (Forrest I-IIb) will become low risk lesions (IIc or III) during this time interval and the majority of rebleeding occurs [7]. Further, the incidence of rebleeding within the first 5 days following treatment of bleeding esophageal varices has been reported up to 40%, so caution within this time frame is felt to be critical [10]. Additionally, should the patient rebleed and require repeat endoscopy or surgery, it was felt best to keep them NPO to not impede visualization. Other efforts to reduce the risk of rebleeding through fasting include emphasis on raising intragastric pH, limiting shear forces, and decreasing splanchnic blood flow.

Feeding and Intragastric pH

Arguments against early feeding for ulcer patients often point to controlling intragastric pH as a means to decrease rebleeding rates [2, 3]. Raising intragastric pH is critical to clot formation and stabilization over ulcers as pepsin, the proteolytic enzyme in the stomach that lyses clots, is inactivated above a pH of 4.5. Since feeding stimulates gastric acid secretion, it was hypothesized that fasting would reduce it. However, the effect of enteral nutrition on intragastric pH is variable [11, 12] likely due to the competing nature of the alkaline enteral formula buffering any increase in gastric acid secretion.

Following the introduction of H_2-receptor antagonists and proton pump inhibitors (PPIs) there was additional concern that feeding could interfere with the acid suppressing effect of these medications and alter their ability to raise intragastric pH. While the use of acid suppressive therapy does raise intragastric pH and is recommended for patients with high risk lesions as it reduces rebleeding rates [13], the effect of feeding patients already on acid suppressive therapy appears to have little influence on intragastric pH [14]. Current standard of care involves high dose intermittent PPI therapy or intravenous bolus followed by continuous infusion for 72 h in patients with bleeding ulcers with high-risk stigmata and continued oral PPI therapy for at least two weeks with a total duration based on the nature of the bleeding lesion [15, 16••]. Perhaps prior to the introduction of acid suppressive

Table 1 Forrest classification and associated risk of rebleeding [2, 6–9]

	Forrest Classification	Endoscopic Findings	Risk of Rebleeding without Endoscopic Therapy (%)	Risk of Rebleeding with Endoscopic Injection (%)
Active Hemorrhage	Ia	Active spurting	65–85	23.6
	Ib	Active oozing	55	19.0
Signs of Recent Hemorrhage	IIa	Non-bleeding visible vessel	43	19.5
	IIb	Adherent clot	22	17.0
	IIc	Flat pigmented spot	10	9.7
Lesions Without Active Bleeding	III	Clean ulcer base	5	1.1

medications, fasting had a role in controlling intragastric pH, but now it seems passé.

Feeding and Shear Forces

The potential for shear forces to dislodge newly formed clot or recently placed clips or bands also contributes to the predilection for fasting. While there are reports of transient dysphagia to solid food following variceal ligation [10, 17••], harmful shear forces arising from food or the presence or placement of a nasoenteric tube is not supported by the current literature [2].

Feeding and Splanchnic Blood Flow

Early management to reduce rebleeding risk also aimed at decreasing splanchnic blood flow. It was hypothesized that early refeeding shifts blood flow to the splanchnic circulation resulting in increased pressure, particularly within varices, and resultant increased risk for rebleeding [2, 3, 10, 12]. Current standard of care includes a combination of variceal ligation, vasoconstrictors, and antibiotics for the treatment of bleeding esophageal varices. It is unclear if the beneficial mechanism of vasoconstrictors is that they reduce the risk of postprandial hyperemia in cirrhotic patients, however vasoconstrictors are known to reduce mesenteric blood flow and therefore may reduce this response [3, 12, 18]. Animal models corroborate that enteral nutrition increases splanchnic blood flow, but they also demonstrate increased gastric motility and improved mucosal integrity with enteral feeds [7]. These findings provide a basis for explaining stress related mucosal damage as a combination of splanchnic hypoperfusion in the presence of gastric acid with prolonged acid exposure [19, 20], and conversely offer a rationale for the benefits of enteral nutrition in preventing stress related mucosal damage.

Clinical Trials of Early Versus Delayed Feeding

Ulcer Related Upper GI Bleeds

Early work by Laine et al. [5] advocated for early refeeding in low risk (Forrest III) non-variceal upper GI bleeds based on etiology and endoscopic evaluation. Subsequent works seek to parse out the timing of refeeding in higher-risk non-variceal bleeds with 4 randomized clinical trials being performed between 1995 and 2020 (Table 2).

Beginning in 1995, Hepworth et al. [21] reported a prospective trial randomizing 95 patients with Forrest Ib or IIa lesions to receive either a prescribed diet or kept NPO for 24 h before transitioning to the prescribed diet. Repeat endoscopies were performed and early feeding did not impede repeat endoscopy. There was no difference in rebleeding rates between groups.

Similarly de Lédinghen et al. [22] randomized Forrest Ib, IIa, and IIb ulcer patients to a prescribed diet progression over 3 days versus keeping them NPO for 3 days prior to beginning their diet progression. Khoshbaten et al. [23] randomized patients with Forrest IIa and IIb ulcers to receive a liquid diet starting at either 6 or 72 h following endoscopy. Both studies found no difference in rebleeding rate between groups, but both studies noted significantly shorter hospital stays in the early feeding group (6.8 ± 2.1 days versus 9.9 ± 3.7 days, $p = 0.01$) and (4.2 ± 1.2 vs. 5.9 ± 1.4 days, $p < 0.001$), respectively. They concluded that there was no advantage to fasting bleeding peptic ulcer patients who were endoscopically treated.

Gong et al. [24••] performed the most recent attempt to evaluate the risk of early refeeding in patients with bleeding peptic ulcers utilizing a noninferiority trial to randomize 218 patients undergoing successful endoscopic hemostasis of bleeding peptic ulcers (Forrest Ia-IIb) to either resume feeding at 24 or 48 h following intervention. Hemostasis was achieved using either thermal coagulation or mechanical therapy with or without injection therapy. Patients in both groups received intravenous pantoprazole or esomeprazole with an initial bolus injection of 80 mg followed by an 8 mg/h infusion prior to endoscopy and transitioned to oral therapy of daily lansoprazole 30 mg, esomeprazole 40 mg, or pantoprazole 40 mg with infusion discontinuation after feeding was initiated. A little over 50% of the population was found to have *Helicobacter pylori* infection although they provided no data on the distribution of these patients specific PPI dosing and duration [25]. More patients in the 24 h group had a history of previous peptic ulcer disease compared to the 48 h group (27.2% versus 10.4%). Thermal coagulation alone was used most commonly to achieve hemostasis (69.9% and 65.1% of cases) and combination therapy was used only 27.2% and 31.1% of the time. Recurrent bleeding rates at 7 days were 7.9% in the 24-h group and 4.0% in the 48 h group and thus noninferiority was not achieved ($p = 0.034$). However, 30-day mortality was lower in the 24 h group than the 48 h group 5.9% versus 14.1% but these deaths were reportedly not related to bleeding events yet not further elaborated on. None of the patients that rebled were Forrest IIb.

Variability in dietary advancement algorithms clouds some interpretation of these studies as does the introduction and subsequent use of acid suppressive therapy and different hemostatic techniques. For instance acid suppressive medication was given at a low dose either daily or twice daily in the de Lédinghen [22] study as these medications first came in to use compared to an initial bolus and continuous infusion in the Gong study [24••] based on an expanding

Table 2 Randomized controlled trials evaluating timing of feeding after bleeding from ulcer disease. NPO, nil per os; PPI, proton pump inhibitor

Author, year N	Forrest class	Diet	Findings	Comments
Hepwroth 1995 95 patients	Forrest Ib or IIa	Normal diet with 50 ml of milk every 2 h VERSUS NPO for 24 h then a normal diet with 50 ml of milk every 2 h	No difference in rebleeding rates.	No difficulty with repeat endoscopy if needed. Adrenaline and ethanolamine sclerotherapy.
de Lédinghen 1998 26 patients	Forrest Ib, IIa, and IIb	Milk on day 1, mixed warm feeds on day 2, and a normal diet on day 3 VERSUS NPO for 3 days, then milk on day 4, mixed warm feeds on day 5, and a normal diet on day 6.	No difference in rebleeding rate. Significantly shorter hospital stay with early feeding.	Adrenaline and ethanolamine sclerotherapy. Used low dose acid suppressive therapy.
Khoshbaten 2013 100 patients	Forrest IIa and IIb	Liquid diet from 6 to 12 h following endoscopy VERSUS NPO until day 3	No difference in rebleeding rate. Significantly shorter hospital stay with early feeding.	Sclerotherapy used in most cases, otherwise argon plasma coagulation.
Gong 2020 218 patients	Forrest Ia, Ib, IIa, IIb	NPO for 24 h then sips of water for 6 h, then liquid diet for 24 h, then soft diet for 24 h, then a regular diet VERSUS NPO for 48 h then sips of water for 6 h, then liquid diet for 24 h, then soft diet for 24 h, then a regular diet	Noninferiority not achieved	Used thermal coagulation or mechanical therapy with or without injection therapy. PPI bolus/infusion. Transition to low dose PPI

body of literature. Oddly, the Gong study has differential durations of PPI infusion therapy for its treatment arms and transitioned patients from infusion to a low dose PPI regimen as opposed to high dose PPI regimen which would otherwise be indicated for these high risk patients [24, 25]. Additionally, guidelines for hemostatic technique [15, 16••] now recommend combination therapy over injection therapy alone, which was the primary means of hemostasis for the earlier studies [21–23]. Further, while a 6.8 versus 9.9 day average hospitalization was acceptable in 1998, care practices have shifted to making an average hospitalization of 4.2 versus 5.9 days the norm by 2013 [22, 23], although specific discharge criteria were not included in every study.

Other areas of inconsistency among these studies include variation in inclusion criteria; most studies specifically excluded Forrest Ia lesions while Gong et al. purposefully included them [21–23, 24••, 25]. Also many patients across these studies underwent second look endoscopy either per protocol or at the provider's discretion and it is unclear what treatments the patients received at the first endoscopy versus the second or the reason that the second endoscopy was performed. [21, 23, 24••, 25] A noted benefit however, is that no study noted feeding to prohibit or complicate repeat endoscopy.

Variceal Upper GI Bleeds

Esophageal varices are a stigmata of cirrhotic liver disease. Gastric varices can represent either extensions of esophageal varices below the gastroesophageal junction, isolated varices related to cirrhosis and portal hypertension, or the result of splenic or portal vein thrombosis. Sources classically recommend holding nutrition support for 48 to 72 h following endoscopic management of esophageal varices given an increased incidence of rebleeding within the first 5 days following therapy, up to 40% [10]. Prior to 2015, there is only one randomized control trial examining timing of nutrition implementation in cirrhotic patients after management of bleeding esophageal varices (Table 3) [26].

De Lédinghen et al. [26] aimed to assess the nutritional and clinical effects of early feeding in cirrhotics following emergency treatment of bleeding esophageal varices. Twenty-two patients were randomized to early enteral nutrition via a nasogastric tube versus no oral diet for 3 days. All patients were then advanced along a progressive diet until day 6. While there was no statistical difference between the groups, 4 of 12 patients in the early feeding group rebled and rebled sooner than the 1 of 10 patients in the delayed feeding group that rebled. Additionally, deaths among patients fed early were interpreted as potential complications associated

Table 3 Randomized controlled trials evaluating timing of feeding after bleeding from variceal disease. NPO, nil per os

Author, year N	Treatment	Diet	Findings	Comments
de Lédinghen 1997 22 patients	Injection sclerotherapy or endoscopic variceal ligation. Continuous octreotide infusion.	Bolus tube feeding from day 1 VERSUS NPO for 3 days then oral diet	No significant difference in rebleeding rate.	No differences in length of stay. Study stopped early due to concern for harm with early feeding.
Lo 2015 70 patients	Endoscopic variceal ligation for esophageal varices; histoacryl injection for gastric varices. Vasoconstrictor (terlipressin or somatostatin) for 3 days. Antibiotics (cefazolin or cefotaxime) for 5 days	NPO for 4 h then a liquid diet for 3 days, a soft diet for 3 days, and a regular diet on day 7 VERSUS NPO for 48 h then a liquid diet for 1 day, soft diet for 3 days and a regular diet on day 7	No significant difference in rebleeding rate. Significantly shorter hospital stay with early feeding.	Included gastric varices. Majority varices medium size. 100% 5-day hemostatic rate.
Goda 2018 90 patients	Endoscopic variceal ligation or injection sclerotherapy. Vasoconstrictor (terlipressin or octreotide) for 3 days. Antibiotics (ceftiazone or cefotaxime) for 5 days.	NPO for 4 h then a liquid diet for 48 h, then a soft diet until a regular diet on day 6 VERSUS NPO for 48 h with PN then a liquid diet for 1 day, a soft diet for 2 days, and a regular diet on day 6	No significant difference in rebleeding rate.	Majority varices large size. Bilharziasis endemic to area. Only 4 patients in each group had sclerotherapy. 100% 5-day hemostatic rate.
Sidhu 2019 101 patients	Endoscopic variceal ligation. Vasoconstrictor (terlipressin) for 3 days. Antibiotics (ceftriaxone) for 5 days.	NPO for 1 h then a liquid diet for 4 h, then a regular diet VERSUS NPO for 4 h then a liquid diet for 24 h, then soft diet for 48 h, then regular diet	No significant difference in rebleeding rate.	Majority varices medium size. Protein and calorie intake significantly better for the first 3 days in the early feeding group. Infection rate significantly reduced in early feeding group on post-hoc analysis.

with enteral feeding (1 rebleed, 1 encephalopathy, 1 pneumonia). Due to the lack of observed nutritional benefit and possible harm caused by early nasogastric tube feeding, the study was terminated early.

More recently 3 trials have sought to evaluate timing of nutrition following treatment of bleeding esophageal varices using a combination of endoscopic therapy, vasoconstrictors, and antibiotics [10, 17••, 27]. Lo et al. randomized 70 cirrhotic patients with esophageal or gastric varices to early- versus delayed-feeding. They found that early feeding with a liquid diet 4 h after successful hemostasis of bleeding esophageal and gastric varices did not impact the rate of rebleeding compared to patients with a delay in liquid diet to 48 h. Unexpectedly, they had a hemostatic rate of 100% at 5 days, which contradicts prior literature and was not anticipated.

Meanwhile, Goda et al. [27] examined outcomes of early- versus late-feeding in 90 patients with acute esophageal variceal bleeding in Egypt. Similar to Lo [10], the early feeding group was started on a liquid diet for the first 48 h after endoscopic therapy while the delayed group was kept NPO during this time period. They too had a 100% hemostatic rate in both groups with no evidence of rebleeding incidents within the first 5 days. Two patients in the early-feeding group had rebleeding: 1 on the 7th day and 1 on the 20th day compared to 1 patient in the delayed-feeding group who had rebleeding on the 10th day.

The most recent study by Sidhu et al. examines early- versus delayed-feeding following esophageal variceal band ligation in 101 cirrhotics [17••]. They had the early-feeding group begin a liquid diet 1 h following endoscopic treatment with advancement to a solid diet after only 4 h while the delayed-feeding group was given liquids for 4 h after treatment before slow advancement to a solid diet after 72 h. There was no significant difference in early or delayed rebleeding rates or mortality between groups. Patients in the early-feeding group had a significantly shorter hospital stay (3.8 ± 1.4 vs. 5.4 ± 1.2, $p < 0.001$) and significantly fewer infections were also identified in the early-feeding group.

Probably the greatest surprise from these studies is the low rate of rebleeding. While rebleeding rates have previously been quoted at 40% in the first 5 days following therapy [10] there were only 3 of 261 patients in the 3 most recent studies who had early rebleeding [10, 17••, 27]. This result suggests either that the patient populations studied were not comparable to those of prior studies, or that the hemostatic techniques have improved. In all 3 studies, the majority of patients were Child-Pugh class B cirrhotics. In the Lo and Sidhu studies, the majority of the patients had slightly enlarged ($\geq$ 6 mm) esophageal varices while the Goda study had a larger proportion of patients with large varices. Small varices ($\leq$ 5 mm) are overall felt to have a very low risk of rebleeding so it is unclear how their inclusion in the Lo and Goda studies effected results [10, 18, 27]. Small varices were not included in the Sidhu study [15]. One hypothesis for the increased incidence of large varices in the Goda study is acknowledgement of the potential coexistence of bilharzial periportal fibrosis in addition to hepatic cirrhosis as bilharziasis is endemic to the region of this study in addition to Hepatitis C which can contribute significantly to portal hypertension [27].

Other important notes include that only patients who could have volitional intake were included in these studies, which may be related the decreased incidence of rebleeding in the patient population as the patients would generally need to be less ill. The 3 most recent studies also include use of primary esophageal band ligation, vasoconstrictors and prophylactic antibiotics as part of their treatment regimens as this practice has become standard of care and makes direct comparison to the de Lédinghen study more difficult.

Stress Ulcer GI Bleeds

The pathophysiology of stress related mucosal damage is thought to be related to relative ischemia. Gastric mucosal hypoperfusion during times of critical illness allows for a localized buildup of lactic acid and associated nitric oxide and superoxide radical production as well as reduction in the release to cytoprotective prostaglandins [3]. Further when blood flow is restored, reperfusion injury can occur.

Stress related mucosal damage can only be diagnosed by endoscopy. While the role of endoscopic therapy is limited in stress related mucosal damage due its diffuse nature of mucosal lesions, endoscopy is necessary to confirm diagnosis and rule out other sources of bleeding. Treatment principles for peptic ulcer disease can also be applied to discrete bleeding sites of stress related ulcers. Once the risk factors for stress related mucosal disease are removed, bleeding tends to be self-limited. There are no specific randomized controlled trials on feeding following diagnosis of stress related mucosal damage.

Lower GI Bleeding

Lower GI bleeding rarely requires withholding enteral nutrition. Prospective study of 235 patients who presented with severe hematochezia, a definitive colonic source for the bleed was found in 75% [8]. The etiologies for colonic bleeds included diverticulosis, ischemia, hemorrhoids, rectal ulcer, polyps, cancer and angiomas. Providing enteral nutrition to these patients rarely affects the risk for rebleeding and withholding EN is usually related to patient preparation for colonoscopy [3]. There are no randomized controlled trials evaluating feeding following diagnosis and treatment of lower GI bleeding.

Reasons to Feed

Positions favoring early feeding point to the potential to shorten hospital length of stay, reduce treatment costs, and decrease associated risk for nosocomial infection [5, 14, 23, 28]. Malnourished patients, particularly those with advanced liver disease and variceal hemorrhage, may not be able to tolerate prolonged fasting which can contribute to further catabolic disadvantage, protein breakdown, and worsening of ascites [18]. Feeding patients earlier shortens the timeline of their hospital stay as it compresses the duration which they are observed NPO and observed eating. This abbreviated diet advancement schedule must be acceptable from a safety standpoint with regard to rebleeding risk prior to implementation. Even after establishing safety, it will likely take a prolonged period of time for it become common practice as even hospitals with early discharge algorithms in place for low risk peptic ulcer patients only discharge 45% of patients early [29].

Additionally, aside from the introduction of acid suppressive therapy, progress has been made with endoscopic interventions. It is now known that combination therapy including epinephrine injection plus a second method of hemostasis reduces the rebleeding rate from peptic ulcer disease [30]. Similarly multimodal therapy in the treatment of esophageal variceal bleeding favoring endoscopic variceal band ligation over sclerotherapy, in addition to vasoconstrictor use and antibiotic therapy has improved outcomes [10]. Data over the course of multiple decades therefore become difficult to interpret given the evolution of standard management.

Lastly, studies surrounding stress related mucosal damage point to feeding as inherent to intestinal healing [3]. Providing enteral nutrition to the gut demands gastric mucosal perfusion and likely represents why enteral nutrition acts as such a potent stress ulcer prophylactic agent. In addition to encouraging blood flow, enteral nutrition can act as a direct source of energy for the gastric tissue, stimulate maintenance of the mucus barrier through prostaglandin, mucus, and bicarbonate secretion and potentially facilitate ulcer healing [3].

Conclusions

Review of these multiple randomized controlled trials and experience suggest that the time to feeding after diagnosis of GI bleeding should be based on patient risk factors associated with disease pathophysiology. Patients with GI bleeding associated with gastric erosions or stress related mucosal damage, gastritis, Mallory-Weiss tear, esophagitis, angiodysplasia, Forrest IIc and III ulcers and lower GI bleeding in general are recommended to resume feeding following endoscopy without delay as these bleeds tend to be self-limited and rarely require intervention.

For higher risk (Forrest Ia-IIb) lesions, data remains mixed on the safety of early refeeding. All trials to date have been relatively small scale and not implemented current standard of care practices using combination endoscopic therapy and high dose PPIs. Until those studies are performed, patients with high risk lesions will likely remain NPO for 1–2 days following endoscopic intervention.

Patients with bleeding esophageal varices who undergo esophageal band ligation, vasoconstrictor therapy, and antibiotic prophylaxis and who are able to have volitional intake can likely resume a clear liquid diet immediately following the procedure although further studies are needed to make a recommendation on timing diet advancement to a soft or regular diet. Timing to advancement to a soft diet should not be delayed beyond 48–72 h following the index procedure however.

Declarations

Disclosures The authors have nothing to disclose.

Human and Animal Rights This article does not contain any studies with human or animal subjects performed by any of the authors.

References

Papers of particular interest, published recently, have been highlighted as:
•• Of major importance

1. Wuerth BA, Rockey DC. Changing epidemiology of upper gastrointestinal Hemorrhage in the last decade: a Nationwide analysis. Dig Dis Sci. 2018;63(5):1286–93.
2. Hébuterne X, Vanbiervliet G. Feeding the patients with upper gastrointestinal bleeding. Curr Opin Clin Nutr Metab Care. 2011;14(2):197–201.
3. McClave SA, Chang WK. When to feed the patient with gastrointestinal bleeding. Nutr Clin Pract. 2005;20(5):544–50.
4. Fonseca J, Meira T, Nunes A, Santos CA. Bleeding and starving: fasting and delayed refeeding after upper gastrointestinal bleeding. Arq Gastroenterol. 2014;51(2):128–32.
5. Laine L, Cohen H, Brodhead J, Cantor D, Garcia F, Mosquera M. Prospective evaluation of immediate versus delayed refeeding and prognostic value of endoscopy in patients with upper gastrointestinal hemorrhage. Gastroenterology. 1992;102(1):314–6.
6. Forrest JA, Finlayson ND, Shearman DJ. Endoscopy in gastrointestinal bleeding. Lancet. 1974;2(7877):394–7.
7. Rassameehiran S, Nugent K, Rakvit A. When should a patient with a Nonvariceal upper gastrointestinal bleed be fed? South Med J. 2015;108(7):419–24.

8. Kovacs TO, Jensen DM. Recent advances in the endoscopic diagnosis and therapy of upper gastrointestinal, small intestinal, and colonic bleeding. Med Clin North Am. 2002;86(6):1319–56.
9. Guglielmi A, Ruzzenente A, Sandri M, Kind R, Lombardo F, Rodella L, et al. Risk assessment and prediction of rebleeding in bleeding gastroduodenal ulcer. Endoscopy. 2002;34(10):778–86.
10. Lo GH, Lin CW, Hsu YC. A controlled trial of early versus delayed feeding following ligation in the control of acute esophageal variceal bleeding. J Chin Med Assoc. 2015;78(11):642–7.
11. MacLaren R, Jarvis CL, Fish DN. Use of enteral nutrition for stress ulcer prophylaxis. Ann Pharmacother. 2001;35(12):1614–23.
12. Guillaume A, Seres DS. Safety of enteral feeding in patients with open abdomen, upper gastrointestinal bleed, and perforation peritonitis. Nutr Clin Pract. 2012;27(4):513–20.
13. Lau JY, Sung JJ, Lee KK, Yung MY, Wong SK, Wu JC, et al. Effect of intravenous omeprazole on recurrent bleeding after endoscopic treatment of bleeding peptic ulcers. N Engl J Med. 2000;343(5):310–6.
14. Ozawa T, Yoshikawa N, Tomita T, Akita Y, Mitamura K. The influence of feeding on gastric acid suppression in helicobacter pylori-positive patients treated with a proton pump inhibitor or an H2-receptor antagonist after bleeding from a gastric ulcer. J Gastroenterol. 2003;38(9):844–8.
15. Laine L, Jensen DM. Management of patients with ulcer bleeding. Am J Gastroenterol. 2012;107(3):345–60 quiz 61.
16.•• Barkun AN, Almadi M, Kuipers EJ, Laine L, Sung J, Tse F et al. Management of Nonvariceal Upper Gastrointestinal Bleeding: Guideline Recommendations From the International Consensus Group. Ann Intern Med. 2019;171(11):805–22. **Current guidelines for management of non-variceal upper gastrointestinal bleeding recommending endoscopic combination therapy for hemostasis and prolonged high dose proton pump inhibitor therapy.**
17.•• Sidhu SS, Goyal O, Singh S, Kishore H, Chhina RS. Early feeding after esophageal variceal band ligation in cirrhotics is safe: Randomized controlled trial. Dig Endosc. 2019;31(6):646–52. **Recent randomized controlled trial with administration of a liquid diet in as little as 1 hour following hemostasis of bleeding esophageal varices with no significant difference in rebleeding rates.**
18. Hou MC. Is there adequate evidence to encourage early feeding in patients with acute esophageal variceal bleeding? J Chin Med Assoc. 2015;78(11):631–2.
19. Ali T, Harty RF. Stress-induced ulcer bleeding in critically ill patients. Gastroenterol Clin N Am. 2009;38(2):245–65.
20. Bardou M, Quenot JP, Barkun A. Stress-related mucosal disease in the critically ill patient. Nat Rev Gastroenterol Hepatol. 2015;12(2):98–107.
21. Hepworth CC, Newton M, Barton S, Gorard D, Swain CP, Burnham WR. Randomised controlled trial of early feeding in patients with bleeding peptic ulcer and a visible vessel. Abstract. Esophageal, Gastr Duodenal Disord. 1995;A113.
22. de Lédinghen V, Beau P, Mannant PR, Ripault MP, Borderie C, Silvain C, et al. When should patients with bleeding peptic ulcer resume oral intake? A randomized controlled study. Gastroenterol Clin Biol. 1998;22(3):282–5.
23. Khoshbaten M, Ghaffarifar S, Jabbar Imani A, Shahnazi T. Effects of early oral feeding on relapse and symptoms of upper gastrointestinal bleeding in peptic ulcer disease. Dig Endosc. 2013;25(2):125–9.
24.•• Gong EJ, Lee SJ, Jun BG, Seo HI, Park JK, Han KH et al. Optimal Timing of Feeding After Endoscopic Hemostasis in Patients With Peptic Ulcer Bleeding: A Randomized, Noninferiority Trial (CRIS KCT0001019). Am J Gastroenterol. 2020;115(4):548–54. **Recent randomized noninferiority trial evaluating early versus delayed feeding among patients with high risk peptic ulcer related bleeding that did not demonstrate noninferiority.**
25. Gajendran M, Perisetti A, Goyal H. Timing of feeding after endoscopic intervention in peptic ulcer disease. Am J Gastroenterol. 2020;115(9):1539–40.
26. de Lédinghen V, Beau P, Mannant PR, Borderie C, Ripault MP, Silvain C, et al. Early feeding or enteral nutrition in patients with cirrhosis after bleeding from esophageal varices? A randomized controlled study. Dig Dis Sci. 1997;42(3):536–41.
27. Goda T, Mokhtar A, Anwar R, Hakim H, Eleraki A. Effect of early versus delayed feeding following emergency endoscopic therapy for acute esophageal variceal bleeding on short-term outcomes. Egypt J Intern Med. 2018;30:110–4.
28. Zhang H, Wang Y, Sun S, Huang X, Tu G, Wang J, et al. Early enteral nutrition versus delayed enteral nutrition in patients with gastrointestinal bleeding: a PRISMA-compliant meta-analysis. Medicine (Baltimore). 2019;98(11):e14864.
29. Trawick EP, Yachimski PS. Management of non-variceal upper gastrointestinal tract hemorrhage: controversies and areas of uncertainty. World J Gastroenterol. 2012;18(11):1159–65.
30. Calvet X, Vergara M, Brullet E, Gisbert JP, Campo R. Addition of a second endoscopic treatment following epinephrine injection improves outcome in high-risk bleeding ulcers. Gastroenterology. 2004;126(2):441–50.

Publisher's Note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.